

Amendments to the Specification:

The following amendments to the specification are made with respect to the substitute specification filed on May 3, 2002.

Please replace the paragraph [0060] with the following amended paragraph:

[0060] **Figures 4A, 4B, and 4C** show the alignment of the Neutrokin-alpha nucleotide sequence (SEQ ID NO:1) determined from the human cDNA clone (HNEDU15) deposited in ATCC No. 97768 with related human cDNA clones of the invention which have been designated HSOAD55 (SEQ ID NO:7), HSLAH84 (SEQ ID NO:8) and HLTBM08 (SEQ ID NO:9).

Please replace the paragraph [0730] with the following amended paragraph:

[0730] In other embodiments, antagonists according to the present invention include soluble forms of Neutrokin-alpha and/or Neutrokin-alphaSV (e.g., fragments of Neutrokin-alpha shown in Figures 1A-B that include the ligand binding domain, TNF conserved domain, and/or extracellular domain of Neutrokin-alpha and/or Neutrokin-alphaSV and fragments of Neutrokin-alphaSV shown in Figures 5A-B that include the ligand binding domain, TNF conserved domain, and/or extracellular domain of Neutrokin-alpha and/or Neutrokin-alphaSV). Such soluble forms of the Neutrokin-alpha and/or Neutrokin-alphaSV, which may be naturally occurring or synthetic, antagonize Neutrokin-alpha and/or Neutrokin-alphaSV mediated signaling by competing with native Neutrokin-alpha and/or Neutrokin-alphaSV for binding to Neutrokin-alpha and/or Neutrokin-alphaSV receptors (e.g., DR5 (See, International Publication No. WO 98/41629), TR10 (See, International Publication No. WO 98/54202), 312C2 (See, International Publication No. WO 98/06842), and TR11, TR11SV1, and TR11SV2 (See, U.S. Application Serial No. 09/176,200, now U.S. Patent No. 6,509,173)), and/or by forming a multimer that may or may not be capable of binding the receptor, but which is incapable of inducing signal transduction. Preferably, these antagonists inhibit Neutrokin-alpha and/or Neutrokin-alphaSV mediated stimulation of lymphocyte (e.g., B-cell) proliferation, differentiation, and/or activation. Antagonists of the present invention also include antibodies specific for TNF-family ligands (e.g., CD30) and Neutrokin-alpha-Fc and/or Neutrokin-alphaSV-Fc fusion proteins.

Please replace the paragraph [0731] with the following amended paragraph:

[0731] By a "TNF-family ligand" is intended naturally occurring, recombinant, and synthetic ligands that are capable of binding to a member of the TNF receptor family and inducing and/or blocking the ligand/receptor signaling pathway. Members of the TNF ligand family include, but are not limited to, TNF-alpha, lymphotoxin-alpha (LT-alpha, also known as TNF-beta), LT-beta (found in complex heterotrimer LT-alpha2-beta), FasL, CD40L, (TNF-gamma (International Publication No. WO 96/14328), AIM-I (International Publication No. WO 97/33899), AIM-II (International Publication No. WO 97/34911), APRIL (J. Exp. Med. 188(6):1185-1190), endokine-alpha (International Publication No. WO 98/07880), neutrokine-alpha (International Publication No. WO 98/18921), CD27L, CD30L, 4-IBBL, OX40L, CD27, CD30, 4-1BB, OX40, and nerve growth factor (NGF). In preferred embodiments, the Neutrokine-alpha and/or Neutrokine-alphaSV TNF-family ligands of the invention are DR5 (See, International Publication No. WO 98/41629), TR10 (See, International Publication No. WO 98/54202), 312C2 (See, International Publication No. WO 98/06842), and TR11, TR11SV1, and TR11SV2 (See, U.S. Application Serial No. 09/176,200, now U.S. Patent No. 6,509,173).